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# GANGRENE OF THE LUNG FOLLOWING ARTIFICIAL PNEUMOTHORAX.

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Gangrene of the lung in pulmonary tuberculosis is a very rare condition. Gangrene of the compressed lung in a patient treated with artificial pneumothorax is exceedingly rare; in fact, no case, to the writer's knowledge, has thus far been reported in literature. The following is a report of such a case, with autopsy.

The patient was an American, aged forty-one years; single; teacher. His father died of pulmonary tuberculosis, aged sixty-nine years.

The patient had had numerous attacks of gonorrhea many years before.

The present illness began with a cold. This he was unable to throw off, and three months later definite physical signs were found, and there were tubercle bacilli in the sputum. On his way north to Saranac Lake he went to the Johns Hopkins Hospital, where the Wassermann and gonorrhea-fixation tests were found negative. On his arrival at Saranac Lake he was not below normal weight, had considerable cough and expectoration, and an afternoon fever of 100° to 101°. The chest examination showed a hyperresonant note over the left side, fair breathing, and coarse rales over the entire front. On the right side posteriorly indefinite rales were heard to the seventh vertebral spine. There was a slight scoliosis, with convexity to the right. The rest of the physical examination was negative.

The urine was normal. The sputum contained many tubercle bacilli, and numerous secondary organisms, chiefly streptococcus and staphylococcus.

No special comment is needed here. The case was an ordinary one of advanced active tuberculosis, involving the greater part of the left lung, and a probable involvement of the upper part of the right. The patient was put to bed and a cough sedative given.

During the next two months he did poorly. The cough became very troublesome and the expectoration increased greatly in amount. The temperature ranged daily between 101° and 102°, with morning remissions. The left chest showed dulness and medium coarse rales over most of the side, with signs of cavitation from the clavicle to the fourth rib. The right chest showed fine rales and deep rhonchi to the seventh rib.

The left lung was disintegrating, and the patient was failing rapidly. With the hope of controlling the advancing process in the left lung, artificial pneumothorax was performed. The history of the seven punctures made is as follows:

Entrance was made in the seventh interspace in the anterior axillary line. The pleural cavity was easily found on the first try. The water manometer read −3 to −11; 350 c.c. of warmed nitrogen gas were injected. Pressure was left at −2 to −6. Two days later 500 c.c. were injected, the pressure being −36 to −9 before and +2 to −1 after the injection. Four days later on entrance the manometer read −3 to −8. About 500 c.c. had flowed in when the patient suddenly gave a sharp cry and seized his side near the needle entrance. He held his breath for a moment and groaned as if in great pain. The pain quickly passed off. The pressure was +2 to −2. This was watched for a few moments, but there were no changes. Apparently there were no ill-effects from this refill.

A week later on entrance the manometer read +3 to −3. This increased pressure was doubtless caused by the presence of a small amount of fluid. At the next operation, the pressure being −3 to −10, 400 c.c. of gas were given and the pressure left at +2 to −2.

Meanwhile there had been no noticeable abatement in the symptoms. Although the patient said he felt much better his cough was very hard and explosive, the expectoration was increased, and the temperature persisted. Breath sounds being heard over the upper part of the left chest it was decided to put a greater pressure upon the lung. Accordingly 800 c.c. were given, the pressure being −5 to −10 before and +6 to +2 after the operation. The patient had no distress although feeling "tight," a sensation that lasted but a few hours.

Two weeks later the fluid was as high as the seventh rib. To satisfy the patient, 200 c.c. of gas were injected and pressure left at +5 to 0. At entrance the manometer showed +1 to −4.

This is the record of the nitrogen injections. It is to be noted that except for the sudden pain during the third operation (considered as a torn adhesion) there was nothing untoward in signs or symptoms caused by the treatment. A small amount of fluid formed, but this is encountered in the majority of cases. The pressures were moderate. The amounts of gas taken were small, but from the signs and symptoms it was judged that the collapse was not complete, and fluid was present which at no time was great enough to be withdrawn.

The patient was too ill to have Roentgen-ray pictures taken. The chest examination, besides the fluid, showed diminished and absent breath sounds over most of the left chest except at the apex and along the vertebral groove. The signs in the right chest remained the same.

His symptoms continued without abatement and his general condition became progressively worse. One afternoon, nine days after the last injection of gas, he suddenly raised a large amount of exceedingly foul, greenish sputa. The next morning, during a coughing spell, he filled five or six sputum boxes with the same putrid matter. He said that it burnt his mouth like fire. The stench was noticeable throughout the house. For nearly three weeks this state of affairs continued. He used about two boxes a day. At times the intensity of the odor diminished. On three occasions he raised from a half to one ounce of blood. Toward the end the expectoration became chocolate colored and watery.

The sputa examined showed mostly pus, enormous numbers of acid-fast bacilli, and also Oïdium albicans, Staphylococcus pyogenes aureus, and a Gram-negative bacillus which produced gas smelling like methane.

No breath sounds were heard on the left side. A metallic tinkle was present. A moderate amount of fluid was made out. There were a few scattered rales on the right side.

The patient sank lower and lower, and finally died three weeks after the first evidence of gangrene of the lung.

Autopsy. Aside from a few scattered fresh tubercles on the surface of the liver and slightly enlarged and partly caseous mesenteric glands, there was nothing abnormal found on the examination of the body except the thorax, where the findings were as follows:

On opening the thorax the heart is found pushed over almost to the midline. The left lung is collapsed and lies against the vertebral column and mediastinum. The pleural cavity contains about one liter of brown, foul-smelling fluid. The collapsed lung occupies about one-third of the left thoracic cavity, being 20 x 9 cm. The division line between the lobes is obliterated. The lung is gray green in color and "mushy." There are adhesions running upward and outward from the apex to the parieties. One adhesion is as large as a finger, and of solid fibrous tissue. The other is small and strand-like. The apex of the lung is very adherent. The base of the lung is bound down to the diaphragm by tremendous adhesions. The lung shows five large cavities opening directly into the pleural cavity. They range in size from 1 x 2 cm. to 7 x 4 cm. Four of these openings occupy the anterior surface of the lung while the fifth and largest is located at the posterior fissure between the lung and parietal pleura The edges of these holes are bevelled and shreds of gangrenous lung tissue are protruding from them. On the parietal pleura, corresponding to the edge of the hole at that place, is a cicatrized ridge as if lung tissue had been torn from visceral pleura which had become adherent to the parietal pleura.

The parietal pleura is 2 to 3 mm. thick. The pleura covering the lung is very dense. Both pleuræ are grayish green in color and covered with slimy exudate.

In removing this lung the parietal pleura was removed also. The lung tissue seen through the various openings is everywhere the same, being composed of grayish green, sponge-like tissue, all evidences of normal lung tissue being destroyed. A section through the apex shows no cavity, but the tissue is more or less solid and mottled and areas of caseation are present.

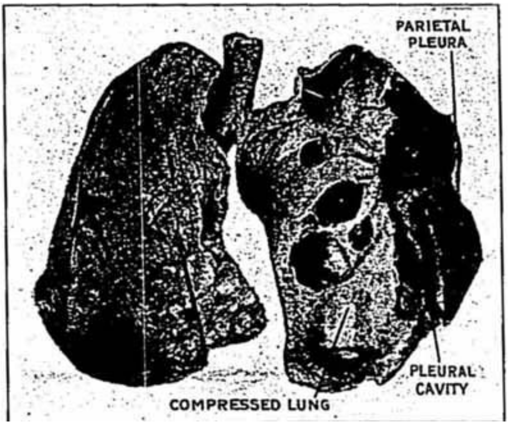
The right or uncollapsed lung is voluminous. The color is bluish pink. There is a fine adhesion at the apex. Over the posterior portion of the lower lobe the pleura is roughened and lusterless. There is no fluid in the pleural cavity. The middle lobe in its central and lower portions shows dense tuberculous areas. On section, scattered tubercles are seen throughout the upper and lower lobes, averaging 1 to 2 cm. in diameter.

The tracheobronchial glands are grayish black in appearance and moderately enlarged, but do not in any way block or narrow the bronchi by pressure. There is no occlusion of the bronchi or bloodvessels entering the lungs.

MICROSCOPIC EXAMINATION. A section taken from the apex of the gangrenous lung shows first a greatly thickened pleura composed of dense connective tissue. Scattered throughout the pleura are numerous minute tubercles. Beneath the pleura no normal lung structure is to be seen. There are small slit-like spaces which are compressed alveoli. Distributed throughout are areas of granulation tissue containing small capillaries in which red blood cells are found. In this granulation tissue are also seen fresh tubercles. Dipping down from the pleura is a dense fibrous band. There are also to be seen large tubercles composed of a central area of fibrous tissue and a dense outer ring of round cells. At the edges of the central area are seen giant cells. The bloodvessels show obliterative endarteritis. Throughout this whole layer there is a dense round-cell infiltration. This layer quickly shades off into an area of non-staining homogeneous tissue in which are scattered portions of nuclei.

Discussion. Was artificial pneumothorax the cause of the gangrene in this case? A brief review of the subject of lung gangrene may help to decide this question.

Gangrene of the lung implies not only death of the tissue but subsequent putrefaction. The necrosis is due to the obstruction of bloodvessels and the deprivation of blood supply. This obstruction may be inflammatory or mechanical. The gangrene is due to the invasion of the necrotic area by putrefactive organisms. Among the organisms found are the pyogenic staphylococcus and streptococcus, the coli communis, and a pseudotubercle bacillus.



Photograph of lungs showing gangrenous compressed left lung with holes opening into pleural cavity through which may be seen the gangrenous tissue.

A frequent direct cause of gangrene is the retention of putrid secretions within a bronchiectatic cavity; also the inhalation of septic material, either during anesthesia or on account of paralysis of the glottis.

Inflammation of the lungs such as pneumonia may lead to gangrene, although some observers doubt its occurrence. It is interesting to note the rarity of gangrene in pulmonary tuberculosis. One would think that bronchial dilatation, caseous areas, and cavities would offer a good field for its occurrence, yet this complication is extremely infrequent. But autopsies show that a localized area of gangrene may exist in a lung without producing fetid sputum or breath, as seen in Fox's series, in which 6 cases of gangrene were found out of 100 postmortems in tuberculous subjects. This is a much higher percentage than other authorities have found. Four of the cases had gangrene in pneumonic areas and two on the walls of cavities.

Tumors of the mediastinum may by pressure lead to gangrenous changes; and septic emboli plugging a pulmonary vessel may cause gangrene. In our case the bronchi and root vessels were patent, and no focus was found at autopsy from which septic emboli could arise.

Occasional cases have been reported in which putrid pleural effusions have been withdrawn, and a fistula being established portions of gangrenous lung have been discharged through the opening. These cases coming to autopsy show the lower portion of the lung compressed by the fluid; the fluid is putrid; and the contiguous lung tissue gangrenous. Loculated fetid empyemas tend to burrow through the lung to a bronchus, and on account of the dense adhesions pneumothorax rarely results. Whether these putrid effusions are primary or secondary is often difficult to say. In the case here reported fluid was noticed after the third refill, but there was nothing in signs or symptoms to lead us to believe that it was gangrenous in character.

Bearing more directly is the question whether gangrene follows collapse or compression of the lung by air as in pneumothorax, or by fluid as in pleurisy with effusion. A review of the literature of many years fails to reveal a single case of gangrene of the lung after a natural or artificial pneumothorax. Evidently the entrance of air into the pleural cavity cannot of itself cause gangrene of the lung.

Fox gives pleurisy with effusion as one of the causes of lung gangrene, but cites no cases and doubts if pressure of fluid in the absence of other factors can produce gangrene. I am not aware of any reports illustrating this condition. The nearest approach to it is found in the cases of putrid effusions or empyemas mentioned above.

Whatever the cause of the obstruction the decomposition of retained secretions is believed by Fowler and Godlee to be the main factor in the production of gangrene of the lung.

In the case here reported there were three factors present which, in combination, were probably sufficient to cause the gangrene. They were (*a*) necrotic areas due to obliterative endarteritis; (*b*) pressure of gas and recoil of lung which aided necrosis, and (*c*) putrefactive organisms.

The following sequence of events might have occurred:

1. To begin with there was a rapidly disintergrating lung with superficial cavities.

2. More or less extensive necrosis of lung tissue due to obliterative endarteritis.

3. The introduction of gas collapsed the lung, limiting the blood supply, and thus aiding in the extension of the necrosis.

4. Putrefaction now took place and gangrene supervened upon the necrotic areas.

5. Rupture of the lung then followed and the pleural fluid became putrid and corroding.

Putrefactive organisms and large necrotic areas are frequently present in tuberculous lungs and do not produce gangrene. There is no reason to suppose that had this lung been let alone it would have become gangrenous. It would appear that the collapsing of the lung was the immediate exciter of the subsequent events.

Other lungs in a similar condition have been collapsed without bad results, but evidently such a procedure is fraught with danger.

I am indebted to Dr. Lawrason Brown for the privilege of reporting this case.

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